Caffeine, performance, and metabolism during repeated Wingate exercise tests

F. GREER, C. MCLEAN, AND T. E. GRAHAM
Department of Human Biology and Nutritional Sciences, University of Guelph, Guelph, Ontario, Canada N1G 2W1

Greer, F., C. McLean, and T. E. Graham. Caffeine, performance, and metabolism during repeated Wingate exercise tests. J. Appl. Physiol. 85(4): 1502–1508, 1998.—Investigations examining the ergogenic and metabolic influence of caffeine during short-term high-intensity exercise are few in number and have produced inconsistent results. This study examined the effects of caffeine on repeated bouts of high-intensity exercise in recreationally active men. Subjects (n = 9) completed four 30-s Wingate (WG) sprints with 4 min of rest between each exercise bout on two separate occasions. One hour before exercise, either placebo (Pl; dextrose) or caffeine (Caf; 6 mg/kg) capsules were ingested. Caf ingestion did not have any effect on power output (peak or average) in the first two WG tests and had a negative effect in the latter two exercise bouts. Plasma epinephrine concentration was significantly increased 60 min after Caf ingestion compared with Pl; however, this treatment effect disappeared once exercise began. Caf ingestion had no significant effect on blood lactate, O2 consumption, or aerobic contribution at any time during the protocol. After the second Wingate test, plasma NH3 concentration increased significantly from the previous WG test and was significantly higher in the Caf trial compared with Pl. These data demonstrate no ergogenic effect of caffeine on power output during repeated bouts of short-term, intense exercise. Furthermore, there was no indication of increased anaerobic metabolism after Caf ingestion with the exception of an increase in NH3 concentration.

ergogenic aids; high-intensity exercise; ammonia; methylxanthines

THE TRADITIONAL HYPOTHESIS for the ergogenic action of caffeine is that it increases catecholamines and fat metabolism, resulting in glycogen sparing (10). If glycogen sparing is the exclusive or critical action associated with caffeine, then its ingestion should have no impact on short-duration exercise when glycogen stores are not limiting. This was demonstrated by Ackman et al. (16), who found that ~50% of glycogen stores remained in the muscle at fatigue during high-intensity exercise [100% maximal oxygen consumption (VO2max)]. The effect of caffeine on short-term (<90 s), supramaximal exercise (i.e., a power output 2–3 times greater than VO2max) has not been well documented, and the performance results are inconsistent. Williams et al. (34) reported no effect on power output or muscular endurance during short-term (15-s) maximal bouts of cycling after caffeine ingestion. A series of investigations produced results ranging from no change in power output (5) to increased peak power (1) and a faster swimming speed (6). Whereas the effects on performance are variable, increases in blood lactate (1, 5–7) and circulating catecholamines (5, 7) have been reported more consistently. On this basis, Collomp et al. (6) hypothesized that the caffeine-induced elevation in epinephrine leads to an increase in anaerobic metabolism resulting in a greater power output during high-intensity exercise. Under these exercise situations, fat metabolism is a minor fuel and muscle glycogen is not limiting. Potential influences of caffeine during short-term high-intensity exercise include a direct effect on skeletal muscle, an impact on excitation-contraction coupling affecting neuromuscular transmission, and an increased mobilization of intracellular calcium from the sarcoplasmic reticulum (28). In support of these possible mechanistic effects of caffeine, Lopes et al. (21) reported that caffeine had a direct effect on skeletal muscle contractile properties. Similarly, Tarnopolsky (28) reported that caffeine delayed fatigue in human muscle during low-frequency stimulation, demonstrating an impact of caffeine on excitation-contraction coupling. Both investigations (21, 28) attributed their results to the effect of caffeine on calcium release from the sarcoplasmic reticulum. Differences in methodology and the difficulty in precisely measuring performance during this type of exercise have led to conflicting results. Well-controlled laboratory investigations are needed to further examine the effect of this methylxanthine on high-intensity exercise.

The purpose of this study was to investigate the effect of caffeine on repeated bouts of high-intensity exercise by using four successive Wingate (WG) tests (WG 1, WG 2, WG 3, and WG 4). Power output and fatigue rate were determined to assess performance. Blood lactate, circulating catecholamines, and respiratory data were collected to determine the effect of caffeine on anaerobic metabolism. It was hypothesized that caffeine ingestion would result in an increase in peak power output, lessen the decline in power output, increase blood lactate and circulating catecholamines, and increase the anaerobic contribution to metabolism.

METHODS

Subjects. Nine healthy men volunteered for the experiment. All were physically active individuals; however, none was accustomed to the intense exercise experienced in this study. The mean age and body weight of the subjects were 29.1 ± 2.7 yr and 82.2 ± 3.5 kg, respectively. The experimental procedures and possible risks were explained to the subjects. Written informed consent was obtained, and the experiment was approved by the ethics committee at the University of Guelph.

Experimental procedure. Each subject reported to the laboratory on four separate occasions. The purpose of the first visit was to familiarize the individual with the equipment and the WG exercise protocol. Visits two and three were the actual WG experiments separated by 1 wk. The purpose of the fourth visit was to obtain steady-state oxygen consumption
(V\textsubscript{O2}) values at several, low exercise intensities. These were determined after 10 min of exercise at a given power output. This was conducted several days after the second WG session. These data were used to predict the metabolic (V\textsubscript{O2}) cost of the power output of the WG test. The calculated V\textsubscript{O2} was then divided by the measured V\textsubscript{O2} and multiplied by 100 to determine the percentage of aerobic contribution of each WG test.

For a given subject, trials 2 and 3 were conducted at the same time of day. The trials were randomized and conducted in a double-blind manner. Subjects were asked to prepare for each testing session in a similar manner and to refrain from caffeine consumption for 48 h before the experiment. On the actual testing day, subjects reported to the laboratory 2–3 h postprandial. At the beginning of each trial, a catheter was placed percutaneously into a medial antecubital vein, and a normal saline drip (0.9% NaCl) was started to maintain catheter patency. A resting blood sample was obtained (~60 min). Subjects then ingested tablets containing either placebo (dextrose) or caffeine (6 mg/kg). This dose of caffeine was chosen because it has been established to elicit an ergogenic effect without approaching the legal limit set by the International Olympic Committee. Furthermore, caffeine doses of 5–6 mg/kg body wt have been shown to saturate the “cytochrome P-450” system in the liver (14). After the subjects rested quietly for 1 h, a second blood sample was taken (0 min). Subsequently, subjects warmed up with light cycling and stretching. An accurate record was kept of the duration and intensity of the warm-up on the first trial and this level was reproduced before the second trial.

The exercise protocol consisted of four 30-s WG sprints with 4 min of rest between each exercise bout. The WG tests were performed on a pan-balance cycle ergometer (Monark 814E Ergomedic, Quinton Instrument, Bothell, WA) with a load of 0.09 kgbody wt (9). The subjects were allowed 5 s of loadless pedaling to reach maximum cadence and were instructed to maintain maximal pedal speed throughout the 30-s period once the appropriate resistance was applied. A computer program was developed in Quick BASIC to measure and record pedal revolutions per minute. An RS232 serial port was used to receive a signal from a switch mounted on the ergometer’s pedal crank to record pedal frequency.

Venous blood samples (7 ml) were obtained before ingestion of capsules and immediately before and after each WG test. The last sample was taken 4 min after the fourth exercise bout. A total of 10 blood samples were obtained.

Calculations. The computerized WG program was used to calculate power output every 5 s for the duration of the test. Average power was calculated as the average of six 5-s power outputs. Peak power was defined as the highest 5-s power output during the 30-s test. Rate of power loss was calculated as (maximum power – minimum power)/30 and was expressed as watts per second.

Analyses.Expired air samples were analyzed for O\textsubscript{2} and CO\textsubscript{2} fractions with an applied O\textsubscript{2} analyzer and a CO\textsubscript{2} detector (Zirconium LB-2 metabolic cart model 2900c, Sensor Medics, Yorba Linda, CA). A breath-by-breath analysis was made of the expired gases, and inspired volume was quantified every 10 s for the duration of the test. The three 10-s V\textsubscript{O2} per minute values of each WG test were summed and divided by 2 to give the total V\textsubscript{O2} for the 30-s test (ml/30 s). Expired volume was determined with a Parkinson-Cowan volumeter. The analyzers were calibrated with gases of known concentrations.

Blood samples (7 ml) were transferred to a sodium-heparinized tube. Hematocrit was measured in duplicate by using high-speed centrifugation. A 200-µl aliquot of blood was added to 1 ml of 0.6 M perchloric acid. A solution of 120 µl of 0.24 M EGTA and reduced glutathione was then added to the remaining blood. Blood samples were spun, and the supernatant was extracted and frozen for future analysis.

The EGTA- and glutathione-treated plasma was analyzed in duplicate for epinephrine (Epi) and norepinephrine (NE) concentrations by HPLC (Waters) as described by Weiker (32). Plasma caffeine, paraxanthine, theophylline, and theobromine were measured by using fully automated HPLC (Waters). One hundred and fifty microliters of plasma were added to ~40 mg ammonium sulfate and 50 µl 0.05% acetic acid. After addition of 25 µl of an internal standard solution (7-β-hydroxypropyl theophylline) and 3 ml chloroform-isopropyl alcohol (85:15, vol/vol) extracting solvent, the mixture was vortexed for 30 s and centrifuged for 10 min at 2,500 rpm. The organic phase was transferred and dried under oxygen-free N\textsubscript{2} and resuspended in HPLC mobile-phase solvent (3% isopropanol, 0.05% acetic acid and 0.5% methanol), and 100 µl were injected into a Beckman, Ultrasphere, IP, C\textsubscript{18}, 5-µl column. Methylxanthines were measured at 282-nm wavelength. Reagents for standards were obtained from Sigma Chemical (St. Louis, MO). The blood-acid extracts were analyzed enzymatically in duplicate for lactate, glucose, and glycerol (3). Plasma samples were analyzed for NH\textsubscript{3} (3) within 1 h of being deproteinized with 0.3 M perchloric acid. Two 1-ml aliquots of whole blood were centrifuged immediately, and the supernate was used to measure potassium within 1 h after the experiment (Ciba-Corning Diagnostics).

Statistics. A two-way ANOVA for repeated measures was used to evaluate statistical significance of the variables measured. A Tukey test was the post hoc analysis used to detect differences over time. Significance was accepted at P ≤ 0.05, and all data are expressed as means ± SE.

RESULTS
Performance. Caffeine ingestion did not have any significant effect on peak power, average power, or the rate of power loss for any of the WG tests. Total power output over the four WG tests was identical in both trials. As expected, peak power output declined with each successive WG test under both treatment conditions. Peak power output in WG1 and 2 was significantly greater compared with WG4 (P < 0.05) (Fig 1). The only significant effect of treatment was in WG4, in which the peak power in the placebo trial was 994 ± 50 W compared with 921 ± 60 W in the caffeine trial (P < 0.05). Average power declined significantly over the first three exercise bouts, with no difference between WG3 and 4 (Table 1). In WG3 and 4, the average power in the placebo trial was greater than that in the caffeine trial. There was no effect of treatment on the rate of power loss (Table 1).

Methylxanthines. Subjects had low levels of caffeine (0.69 ± 0.41 µM) before each trial, confirming their compliance to abstain from methylxanthine-containing products. Sixty minutes after caffeine ingestion (6 mg/kg), plasma caffeine levels were 33.88 ± 4.20 µM.

Catecholamines. Plasma Epi concentrations were significantly elevated 60 min after the ingestion of caffeine (0.62 ± 0.08 nM) vs. placebo (0.27 ± 0.01 nM). However, this treatment effect disappeared once exercise began (Fig 2). There was no effect of caffeine ingestion on NE levels compared with placebo 60 min postingestion. There was a significant increase in NE by the end of WG 4 in the caffeine trial compared with...
placebo. NE concentration increased significantly during the WG tests over resting levels (−60 and 0 min; Fig. 3).

Blood glucose. There was no effect of treatment on blood glucose concentration at any time during the protocol (Table 2).

Glycerol. With each successive WG test there was a significant increase in blood glycerol concentration (Fig. 4). Glycerol concentrations increased fivefold from rest to the end of the exercise period [4 min after WG 4 (R 4)] in the caffeine trial and fourfold in the placebo trial. By the end of the exercise protocol (R 4), glycerol concentration in the caffeine trial was significantly higher compared with placebo.

NH$_3$. After WG 2, plasma NH$_3$ concentration increased significantly with each successive exercise bout (Fig. 5). This increase was significantly higher in the caffeine trial compared with placebo with the exception of one time point [4 min after WG 2 (R 2)].

K$^+$. There was no difference in plasma K$^+$ levels between the caffeine trial compared with placebo. In both trials there was a significant increase in plasma K$^+$ immediately after each WG test, which returned to levels below resting values by the end of the corresponding rest period (Table 2).

Blood lactate. There was no effect of treatment on blood lactate concentration during the exercise session. Blood lactate levels were significantly elevated after WG 1 compared with resting values and continued to increase to the end of WG 2. In WG 3 and 4, blood lactate concentrations were significantly higher than those at rest and in the preceding two WG tests. By the end of the exercise protocol, blood lactate was ~10-fold higher compared with resting values (Table 2).

Respiratory data. Caffeine had no effect on $\dot{V}O_2$ or aerobic contribution during any time throughout the exercise protocol (Table 3). $\dot{V}O_2$ and aerobic contribution increased significantly in WG 2, 3, and 4 regardless of treatment.

Table 1. Summary of performance data

<table>
<thead>
<tr>
<th></th>
<th>WG 1</th>
<th>WG 2</th>
<th>WG 3</th>
<th>WG 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average power, W</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>768 ± 54$^a$</td>
<td>678 ± 26$^a$</td>
<td>633 ± 32$^a$</td>
<td>614 ± 30$^a$</td>
</tr>
<tr>
<td>Caf</td>
<td>777 ± 48$^a$</td>
<td>682 ± 36$^a$</td>
<td>606 ± 33$^a$</td>
<td>599 ± 62$^a$</td>
</tr>
<tr>
<td>Power loss, W/s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>19.3 ± 2.0$^a$</td>
<td>20.1 ± 1.5$^a$</td>
<td>19.7 ± 2.0$^a$</td>
<td>18.7 ± 2.5$^a$</td>
</tr>
<tr>
<td>Caf</td>
<td>20.4 ± 2.3$^a$</td>
<td>20.3 ± 1.8$^a$</td>
<td>20.4 ± 1.8$^a$</td>
<td>16.6 ± 2.8$^a$</td>
</tr>
</tbody>
</table>

Values are means ± SE. Treatment is placebo (PI) or caffeine (Caf). WG 1, 2, 3, and 4, Wingate tests 1, 2, 3, and 4, respectively. Values having the same letter are not significantly different from each other (P > 0.05).
which noted a decline in average power output during exercise bouts is similar to two previously reported studies, decline in power output with successive intense exercise. The average power output in WG 4 and Collomp et al. (6) but are in contrast to findings by Anselme et al. (1) those reported by Williams et al. (34) and Collomp et al. metabolism. The performance results are consistent with high-intensity exercise due to increased anaerobic metabolism, or the aerobic contribution at any point during the exercise protocol. Hence, our results do not support our hypothesis that caffeine ingestion would result in an increase in power output during repeated bouts of high-intensity exercise due to increased anaerobic metabolism. The performance results are consistent with those reported by Williams et al. (34) and Collomp et al. (5) but are in contrast to findings by Anselme et al. (1) and Collomp et al. (6).

There was a significant decrease in average power output over the first three WG tests, with no difference between WG 3 and 4. The average power output in WG 4 was 77% of the power output in WG 1. This trend for a decline in power output with successive intense exercise bouts is similar to two previously reported studies, which noted a decline in average power output during the final exercise bout of 60 and 65% compared with the first exercise session (23, 27).

Caffeine ingestion has been shown to increase plasma Epinephrine levels, both at rest and during prolonged exercise, with little effect on NE concentration (13, 14). Only two investigations examining the effects of caffeine ingestion on high-intensity exercise have measured plasma catecholamines, and they reported elevated levels of Epinephrine and NE (5, 7). On the basis of these data, together with an increase in blood lactate, those studies suggested that the caffeine-induced increase in Epinephrine results in an increase in anaerobic metabolism, promoting a greater power output. The recent finding of Vergauwen et al. (31), which demonstrated an increase in glycogenolysis in contracting rodent muscle after caffeine administration, supports this hypothesis. Nevertheless, our data fail to support this. There was no increase in performance and no increase in lactate or aerobic contribution in the caffeine trial. The effect reported by Vergauwen et al. was predominantly in slow-twitch fibers. However, in this type of intense exercise, fast-twitch muscle fibers probably dominate the metabolic events.

In the present study, plasma Epinephrine concentration increased significantly 1 h after caffeine ingestion compared with placebo. However, this treatment effect
disappeared during exercise and had no effect on power output or anaerobic metabolism. This is consistent with Chesley et al. (4), who reported no effect on glycolysis or lactate concentration in active muscle during Epi infusions that resulted in Epi concentrations comparable to those created by caffeine ingestion.

There was no effect of treatment on blood lactate levels at any time throughout the exercise protocol. Blood lactate concentration increased significantly with each successive work bout until WG 3, despite a decrease in power output. Although there was a significant difference in blood lactate concentration between WG 2 and 3, the mean change was less compared with the sharp increase that occurred between WG 1 and 2. These results follow the same pattern as those of McCartney et al. (23) and Spriet et al. (27), who reported a reduction in lactate formation in the latter bouts of repeated exercise. It has been suggested that the decline in power output with successive bouts of exercise is accompanied by a reduction in glycolysis and muscle lactate accumulation (23). The plateau of blood lactate concentration may be due to a reduction in anaerobic glycolysis mediated by a downregulation of glycogen phosphorylase activity as a result of changes in H⁺ concentration or Ca²⁺ concentration (27).

The decline in power output and corresponding plateau in blood lactate concentrations with successive exercise bouts were accompanied by an increase in VO₂. In the last three WG trials, VO₂ increased by 23–29% compared with WG 1. Although lactate production and release were not measured in this study, the data are consistent with the hypothesis that lactate production was less during the latter exercise bouts. Because the ATP yield per mole of glucose oxidized is 12 times higher compared with the ATP yield per mole of lactate produced, a small increase in VO₂ would mean a large reduction in ATP required from anaerobic glycolysis. This would lead to a large decrease in lactate production and is consistent with results reported by Bangsbo et al. (2), who found that an initial bout of intense exercise had an inhibitory effect on lactate production in a successive exercise bout.

Trump et al. (29) noted that, with repeated bouts of cycling, ATP resynthesis from anaerobic glycolysis decreased and phosphocreatine and oxidative metabolism become more important sources of ATP. They suggested that, by the third bout of intense exercise, aerobic metabolism accounts for 70% of the total energy provision. Possible sources include free glucose from the muscle and free fatty acid oxidation (23). In the present study, glycerol increased over 400% in the placebo trial and 500% in the caffeine trial. This is similar to the progressive rise in plasma glycerol concentration reported by McCartney et al. (23), who used a similar protocol. The present results support the suggestion by McCartney et al. that intramuscular triglyceride stores likely contribute to the energy requirements of the repeated exercise bouts despite the high intensity. The increase in Epi and NE concentration with each successive WG may have resulted in an increase of lipolysis. In support of this, there was an obligatory increase in VO₂ in the latter exercise bouts.

During intense exercise such as in the present investigation, NH₃ is produced predominantly via the AMP deaminase reaction with no contribution from the breakdown of amino acids (11, 18). Furthermore, AMP deamination and IMP accumulation occur to a greater extent in fast-twitch fibers compared with slow-twitch muscle fibers (24). The increase in plasma NH₃ concentration during intense exercise has been suggested to reflect the energy state of the cell (26). For our data to support this hypothesis, we would have expected a plateau in plasma NH₃ concentration in WG 3 and 4 because the blood lactate and VO₂ data indicated a greater reliance on aerobic metabolism in these latter work bouts. However, in the present study, lactate increased in the initial WG with no concomitant increase in NH₃, whereas, in the later work bouts, NH₃ increased without a significant rise in lactate levels. This dissociation between lactate and NH₃ has been reported previously by Graham et al. in steady state (12) and intense exercise (11). Furthermore, plasma NH₃ concentration was higher from WG 2 to the end of R 4 with the exception of one data point (R 2) in the caffeine trial compared with placebo. From these data, it appears that caffeine results in an increase in plasma NH₃ concentrations but not lactate. We have no explanation for rise in NH₃ concentration except to hypothesize that methylxanthine ingestion may alter either the handling of ADP or that of adenosine resulting in a greater production or decreased removal of plasma NH₃.

An increase in contractile activity typically leads to an increase in plasma K⁺ concentration, reflecting a net loss of K⁺ from contracting muscle. An increase in the activation of sarcolemmal Na⁺-K⁺-ATPase, leading to the reuptake of K⁺ into resting muscle and other tissues, has been suggested as a probable explanation (17, 20). In the present study, plasma K⁺ concentration was significantly elevated after each exercise bout and rapidly fell to or below rest concentrations. Although our absolute values were lower compared with those reported by Lindinger et al. (19) during repeated bouts of maximal exercise, the pattern of response is similar.

In the present study, caffeine ingestion had no effect on plasma K⁺ concentration during exercise or in recovery. Lindinger et al. (19) reported an attenuation of an exercise-induced increase in plasma K⁺ uptake by resting muscle after caffeine ingestion during pro-
longed submaximal exercise. The negligible effect of caffeine in the present study may be due to the short time frame of the exercise and recovery bouts. Therefore, even if caffeine is stimulating the uptake of K⁺ by other muscles, detection may not be possible because the magnitude of the K⁺ flux may already be maximal. Furthermore, sampling times in the present study do not allow us to assess the effect of caffeine on the reuptake of K⁺ early in the recovery process.

The effects of caffeine on blood glucose responses to high-intensity intermittent exercise have not been previously reported. Although not statistically significant, blood glucose concentration was higher immediately after WG 4 and 4 min into recovery in the caffeine trial compared to placebo, suggesting that caffeine may lead to a reduction in glucose uptake resulting in elevated blood glucose levels. This would support the earlier finding by Vergauwen et al. (30), who reported that a physiological concentration of caffeine resulted in a 20–25% decrease in glucose uptake in a rodent contractile hindlimb perfusion.

There is little doubt that caffeine results in an increase in exercise time to exhaustion during prolonged, submaximal exercise and even during exercise bouts lasting 4–6 min (16, 33). However, caffeine does not always appear to lead to an increase in performance in sprint-type exercise or in repeated bouts of intense exercise and may in fact have a negative effect. The reasons behind this are unclear. The dose used in the present study (6 mg/kg) has been shown to be effective ergogenically during submaximal (8, 13, 22) and maximal exercise (16). Several mechanisms of caffeine demonstrated in in vitro studies that used higher levels of this substance include a caffeine-induced Ca²⁺ release from the sarcoplasmic reticulum, a direct effect on myofilaments, an inhibition of phosphodiesterase and alterations in neuromuscular transmission (28). It is possible that caffeine may influence muscle function through several of these mechanisms, and therefore a higher dose of caffeine may be needed to elicit an ergogenic effect.

The training status of the individual may also be important. In the present study, subjects were recreationally active; however, none were specifically anaerobically trained athletes. Furthermore, the recruitment of fast-twitch fibers would be significant in the type of exercise used in the present investigation. It is unlikely that motor unit activation is 100% in untrained individuals. If caffeine is exerting an ergogenic effect through fast-twitch fibers, the “signal” may result in an enhanced response early in the protocol yet lead to a greater decrease with successive work bouts.

It is also possible that caffeine’s effect may be elicited primarily in oxidative muscle fiber types. An increased sensitivity to caffeine has been demonstrated in slow-twitch fibers in humans (25). Consistent with this, Vergauwen et al. (30, 31) were only able to show a caffeine effect on carbohydrate metabolism in slow-twitch fibers during electrical stimulation in the rodent, and they attributed this effect to adenosine-receptor antagonism. Preliminary evidence exists suggesting adenosine (A₂) receptors exist only in slow-twitch, oxidative muscle fibers (15). Therefore, if adenosine-receptor antagonism is one mechanism through which caffeine exerts its ergogenic effect, an increase in performance in high-intensity exercise would not be expected.

Caffeine has been suggested to change central nervous system motor unit recruitment and has been attributed to an increase in calcium mobilization from the sarcoplasmic reticulum (21, 28). Jackman et al. (16) reported a significant increase in muscle lactate production in exercise intensity equivalent to 100% VO₂max after caffeine ingestion. Although speculative, it is possible that the high muscle lactate concentration likely produced in the present protocol could nullify any ergogenic effect of caffeine because lactate may inhibit calcium and caffeine-stimulated calcium release (28).

From the present study, caffeine has no beneficial effect on peak power and may have a negative effect in later work bouts. Caffeine ingestion resulted in an increase in E₁p levels only at rest, with no effect on blood lactate concentration or VO₂ at any time throughout the protocol. Thus there is no indication of increased anaerobic metabolism after caffeine ingestion, with the exception of an increase in NH₃ concentration.

The authors acknowledge the excellent technical support of P. Sathasivam. This study was supported by research grants from the National Science and Engineering Research Council of Canada and Sport Canada.

Address for reprint requests: T. E. Graham, Dept. of Human Biology and Nutritional Sciences, Univ. of Guelph, Guelph, ON, Canada N1G 2W1 (E-mail: tgraham.ns@aps.uoguelph.ca).

Received 10 December 1997; accepted in final form 31 June 1998.

REFERENCES